Adipocyte 3:3, 167–169; July/August/September 2014; © 2014 Landes Bioscience

In this issue of *Adipocyte*

Azaam M Samad

San Diego Biomedical Research Institute; San Diego, CA USA

Effects of Environmental Pollution on Adipogenesis pp 170–9

Is it possible that the exposure to environmental pollutants may be contributing to the ongoing obesity epidemic? In this research paper, authors Atlas et al. explore the effects of the widely used substance bisphenol A (BPA) on the 3T3L1 cell model without the presence of glucocorticoids (which are known to enhance adipocyte differentiation). The study shows an increase in 3T3L1 cell differentiation in the presence of BPA, in addition to an upregulation of mRNA expression and protein levels in the terminal marker of adipogenesis aP2 (**Fig. 1**).

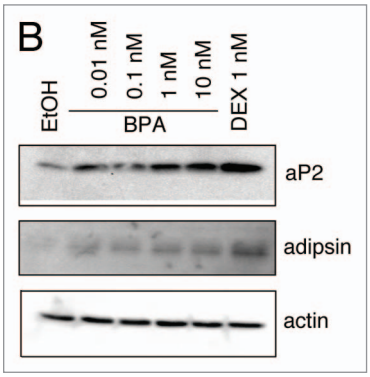

Figure 1. Figure detail from Atlas et al., p 171.

Visfatin and TNFα-Mediated Insulin Resistance pp 180–9

As authors Gouranton et al. explain in this research paper, TNFα is seen as a disruptor of insulin signaling and, recently it has been shown that visfatin plays a crucial role as well. However, whether there is a link between the two pathways is unknown. This study attempts to clarify the role of visfatin with regards to TNFα's impairment of the insulin pathway. The authors show that visfatin is downgraded in 3T3L1 cells after TNFα treatment, and link an increase in protein-tyrosine phosphate 1B (PTP1B) expression to this downregulation via decreases of nicotinamide adenine dinucleotide concentrations followed by decreases in Sirtuin 1 activity. The increase in PTP1B by visfatin likely contributed to the observed increase in insulin resistance in these adipocytes.

PCOS, Insulin Resistance, and Adipose Dysfunction pp 190–6

PCOS, or Polycystic Ovary Syndrome, is associated with numerous type 2 diabetes risk factors, including insulin resistance, obesity, and low-grade inflammation. It is thought that insulin resistance is one of the biggest contributors to the disturbances present in PCOS. Could it be that adipose tissue dysfunction is a key factor in this insulin resistance? Authors Mannerås-Holm et al. show, through a study of 44 genes, that women with PCOS and control groups showed novel differences in adipose tissue mRNA expression of genes involved in the pathogenesis of PCOS and adipose dysfunction.

Low In Vitro Subcutaneous Adipogenesis is Associated with Visceral Obesity and Metabolic Dysfunction pp 197–205

Although research has shown that adipogenic capacity appears to be reduced in obesity, there is no known study that attempts to shed light on in vitro differentiation of primary preadipocytes from various fat compartments and its relation to visceral obesity. Using adipose tissues samples from women undergoing gynecological surgery authors Lessard et al. relate in vitro adipogenesis to body fat distribution, cell size in the tissue and metabolic dysfunction. In this research paper the authors present their findings and show that there is an association between visceral obesity, metabolic abnormalities and low abdominal subcutaneous preadipocyte differentiation capacity in vitro.

Cre Recombinase Models for Adipocytes and Adipocyte Precursors pp 206–11

Genetic mouse models such as the Cre/lox system of gene targeting have advanced the study of adipose tissue in vivo greatly, with aP2-Cre^{BI} and aP2-Cre^{Salk} lines being prolifically used in the targeting of adipose tissue. Here, authors Jeffery et al. look into the specificity and efficiency of Cre recombinase activity for adipocytes within adipose tissue. Their research shows a lack of specificity for adipocytes with regards to aP2-Cre^{BI} and aP2-Cre^{Salk} lines, and inefficient labeling of adipocytes with the aP2-CreBI line. The Adiponectin-CreERT was however highly efficient and specific in targeting adipocytes. The authors also discuss their findings using the PdgfRα-Cre line, suggesting that it is currently the most preferable model for in vivo studies of adipocyte precursor cells (**Fig. 2**).

Improvements through Tempering pp 212–4

Lipocryolysis (a localized fat reduction treatment) combines a vacuum with

Correspondence to: Azaam M Samad; Email: asamad@sdbri.org
Submitted: 07/18/2014; Accepted: 07/18/2014; Published Online: 07/18/2014
http://dx.doi.org/10.4161/adip.30000

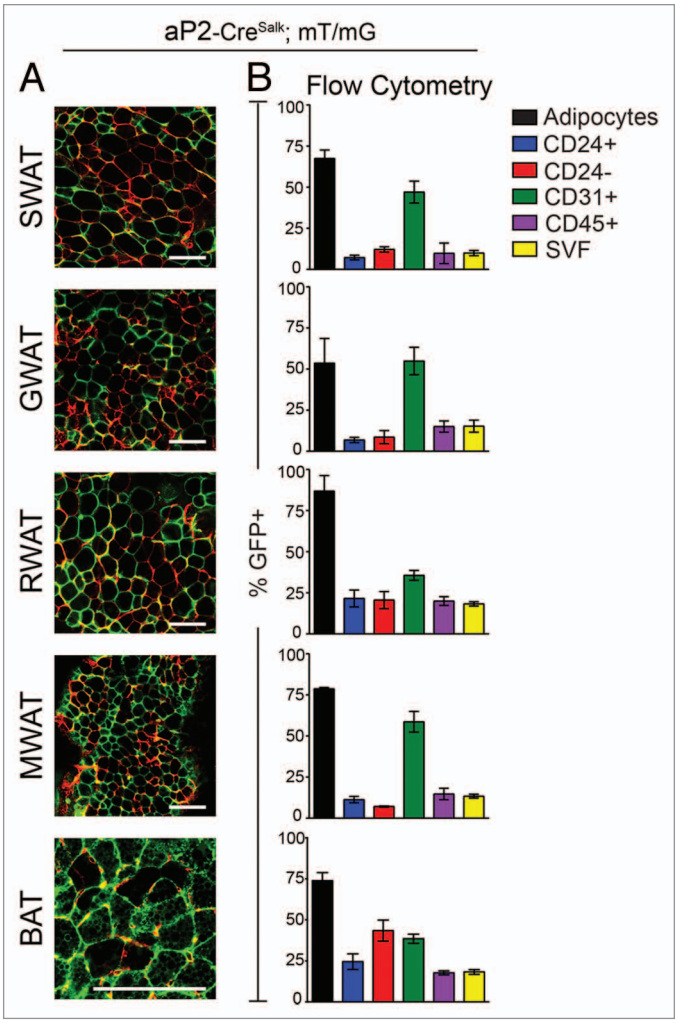

Figure 2. Figure from Jeffery et al., p 208.

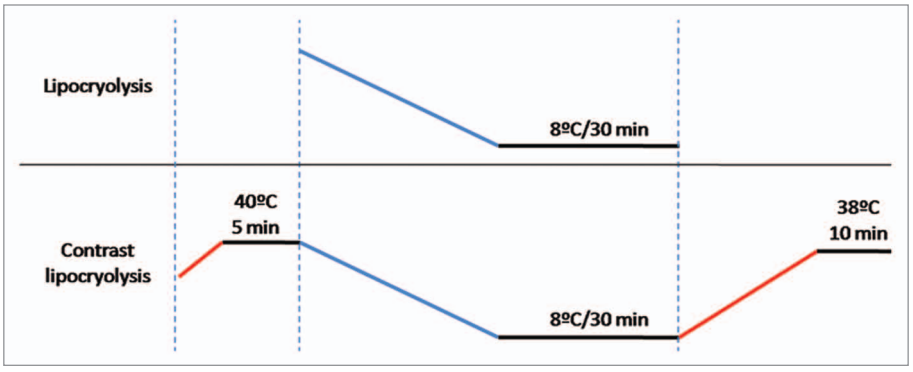

Figure 3. Figure from Pinto and Melamed, p 214.

adipose tissue heat extraction. Advances in research and technology have yielded further insights with regards to various aspects of lipocryolysis, and "tempering technology" developed in the food industry to temper lipids combined with lipocryolysis has led to "contrast lipocryolysis'" technology. This brief report by authors Pinto and Melamed studies the results of 10 contrast lipocryolysis subjects after a single session and found a 42.45% improvement in fat layer reduction when compared with conventional lipocryolysis (**Fig. 3**).

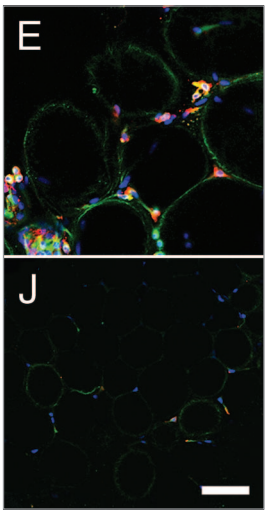

Figure 4. Figure detail from Buckman et al., p 217.

White Adipose S100B Levels and the Link to Obesity-Associated Inflammation pp 215–20

Authors Buckman et al. seek to understand more about the distribution and physiological regulation of the calcium binding protein S100B in this brief report. The study looked at plasma S100B concentrations as well as white adipose tissue S100B mRNA levels in lean and diet-induced mice, and found both levels to be increased by obesity. In the case of white adipose tissue S100B levels, the noted increase dropped after weight-loss. These findings support a link between white adipose tissue S100B and obesity-associated inflammation (**Fig. 4**).

Thermogenesis through Low Body Temperature? pp 221–3

Energy expenditure through thermogenesis via the activation of brown adipose tissue is the current focus of efforts to treat obesity and associated disorders. Authors Warner and Mittag attempt to show a different perspective in this commentary. Inspired by their own recent study

involving a defective thyroid hormone signaling and its link between a lower body temperature and elevated brown adipose tissue thermogenesis, this commentary presents a new avenue of study for the field of thermogenesis to explore (**Fig. 5**).

Macrophage Activation and Obesity-Induced Oxidative Stress pp 224–9

Authors Frohnert and Bernlohr recently demonstrated that inflammatory changes in macrophages were induced by products of lipid peroxidation such as glutathionyl-4-hydroxy-2-nonenal (GS-HNE) and glutathionyl-1,4-dihydroxynonene (GS-DHN) from adipocytes. An increase in fasting glucose levels and impaired glucose tolerance was also found to be associated with overproduction of GS-HNE. This commentary discusses the links between obesity-induced oxidative stress in adipocytes and the activation of tissue resident macrophages, a pathway which merits further study for the benefit of combating the metabolic effects of obesity.

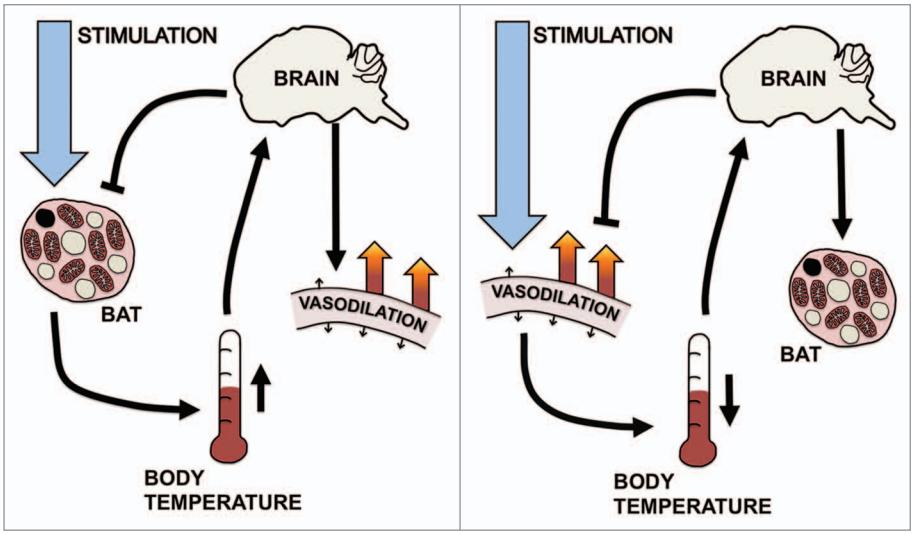

Figure 5. Figure from Warner and Mittag, p 223.